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NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	AUG 10	Time limit for inactive STN sessions doubles to 40 minutes
NEWS	3	AUG 18	COMPENDEX indexing changed for the Corporate Source (CS) field
NEWS	4	AUG 24	ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced
NEWS	5	AUG 24	CA/CAplus enhanced with legal status information for U.S. patents
NEWS	6	SEP 09	50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY
NEWS	7	SEP 11	WPIDS, WPINDEX, and WPIX now include Japanese FTERM thesaurus
NEWS	8	OCT 21	Derwent World Patents Index Coverage of Indian and Taiwanese Content Expanded
NEWS	9	OCT 21	Derwent World Patents Index enhanced with human translated claims for Chinese Applications and Utility Models
NEWS	10	NOV 23	Addition of SCAN format to selected STN databases
NEWS	11	NOV 23	Annual Reload of IFI Databases
NEWS	12	DEC 01	FRFULL Content and Search Enhancements
NEWS	13	DEC 01	DGENE, USGENE, and PCTGEN: new percent identity feature for sorting BLAST answer sets
NEWS	14	DEC 02	Derwent World Patent Index: Japanese FI-TERM thesaurus added
NEWS	15	DEC 02	PCTGEN enhanced with patent family and legal status display data from INPADOCDB
NEWS	16	DEC 02	USGENE: Enhanced coverage of bibliographic and sequence information

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FULL ESTIMATED COST                                 0.22       0.22

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=> s (G(w)CSF or granulocyte(w)colony(w)stimulating(w)factor)
L1       76986 (G(W) CSF OR GRANULOCYTE(W) COLONY(W) STIMULATING(W) FACTOR)

=> s l1 and (traumatic(w)brain(w)injury)
L2          35 L1 AND (TRAUMATIC(W) BRAIN(W) INJURY)

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3           24 DUP REM L2 (11 DUPLICATES REMOVED)

=> dis ibib abs l3 1-24

L3   ANSWER 1 OF 24  EMBASE  COPYRIGHT (c) 2009 Elsevier B.V. All rights
     reserved on STN
ACCESSION NUMBER:   2009329194  EMBASE
TITLE:              Pharmacotherapy in stroke rehabilitation.
AUTHOR:             Czlonkowska, Anna, Dr. (correspondence)
CORPORATE SOURCE:   Institute of Psychiatry and Neurology, 2nd Dept of
                    Neurology, 9 Sobieskiego Str., 02-957 Warsaw, Poland.
                    czlonkow@ipin.edu.pl
AUTHOR:             Czlonkowska, Anna, Dr. (correspondence); Lesniak, Marcin
CORPORATE SOURCE:   Medical University, Department of Clinical Pharmacology,
                    Warsaw, Poland. czlonkow@ipin.edu.pl
SOURCE:             Expert Opinion on Pharmacotherapy, (June 2009) Vol. 10, No.
                    8, pp. 1249-1259.
                    Refs: 95
                    ISSN: 1465-6566  CODEN: EOPHF7
PUBLISHER:          Informa Healthcare, Telephone House, 69 - 77 Paul Street,
                    EC2A 4LQ, United Kingdom.
COUNTRY:            United Kingdom
DOCUMENT TYPE:      Journal; General Review; (Review)
FILE SEGMENT:       008     Neurology and Neurosurgery
                    019     Rehabilitation and Physical Medicine
                    030     Clinical and Experimental Pharmacology
                    037     Drug Literature Index
                    038     Adverse Reactions Titles
LANGUAGE:           English
SUMMARY LANGUAGE:   English
ENTRY DATE:         Entered STN: 18 Aug 2009
                    Last Updated on STN: 18 Aug 2009
AB   Background: Pharmacotherapy is commonly given to patients recovering from
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a stroke to prevent further complications (e.g. recurrent stroke, seizures) or enhance recovery. However, some drugs may have a negative impact on neuroplasticity. Objectives: This review examines currently used drugs that are believed to promote recovery from motor and cognitive disturbances associated with stroke. Methods: Literature regarding the properties, efficacy, safety, and dosing of drugs used to promote recovery after stroke was reviewed. Results: The data on pharmacotherapy are insufficient to support a claim of significantly improved rehabilitation outcomes. Moreover, a growing body of evidence indicates that some agents can impair functional reorganization and slow the recovery process. However, a few chemicals are reported to be beneficial for stroke rehabilitation. The most promising are noradrenergic and dopaminergic agents, as well as several growth factors; these should be the future focus of extensive randomized clinical trials. Conclusions: Currently there is no drug with proven efficacy in enhancing poststroke recovery.

L3 ANSWER 2 OF 24 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2009:618396 BIOSIS
DOCUMENT NUMBER: PREV200900619499
TITLE: Combination therapy for traumatic brain injury in rats with stem cell mobilization by granulocyte-colony stimulating factor and umbilical cord matrix stem cell injection enhance recovery.
AUTHOR(S): Modiry, N. [Reprint Author]; Marzban, M.; Ebrahimi, A.
CORPORATE SOURCE: Iran Univ Med Sci, Tehran, Iran
SOURCE: European Journal of Neurology, (OCT 2009) Vol. 16, No. Suppl. 3, pp. 597.
Meeting Info.: 13th Congress of the European-Federation-of-Neurological-Societies. Florence, ITALY. September 12 -15, 2009. European Federat Neurol Soc.
ISSN: 1351-5101.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Nov 2009
Last Updated on STN: 12 Nov 2009

L3 ANSWER 3 OF 24 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
ACCESSION NUMBER: 2009:618395 BIOSIS
DOCUMENT NUMBER: PREV200900619498
TITLE: Mobilization of stem cell with granulocyte-colony stimulating factor promotes recovery after traumatic brain injury in rat.
AUTHOR(S): Marzban, M. [Reprint Author]; Modiry, N.; Ebrahimi, A.
CORPORATE SOURCE: Iran Univ Med Sci, Tehran, Iran
SOURCE: European Journal of Neurology, (OCT 2009) Vol. 16, No. Suppl. 3, pp. 596.
Meeting Info.: 13th Congress of the European-Federation-of-Neurological-Societies. Florence, ITALY. September 12 -15, 2009. European Federat Neurol Soc.
ISSN: 1351-5101.
DOCUMENT TYPE: Conference; (Meeting)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Nov 2009
Last Updated on STN: 12 Nov 2009

L3 ANSWER 4 OF 24 EMBASE

ACCESSION NUMBER: 2008604262 EMBASE
TITLE: Therapeutic Potential of Erythropoietin and its Structural or Functional Variants in the Nervous System.
AUTHOR: Siren, Anna-Leena (correspondence); Fasshauer, Theresa
CORPORATE SOURCE: Department of Neurosurgery, University of Wurzburg, Wurzburg, 97080, Germany. siren.a@nch.uni-wuerzburg.de
AUTHOR: Bartels, Claudia; Ehrenreich, Hannelore
CORPORATE SOURCE: Division of Clinical Neuroscience, Max-Planck-Institute of Experimental Medicine, Gottingen, 37075, Germany. ehrenreich@em.mpg.de
SOURCE: Neurotherapeutics, (January 2009) Vol. 6, No. 1, pp. 108-127.
Refs: 135
ISSN: 1933-7213
PUBLISHER: Elsevier Inc., 360 Park Avenue South, New York, NY 10010, United States.
PUBLISHER IDENT.: S 1933-7213(08)00237-7
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 008 Neurology and Neurosurgery
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
038 Adverse Reactions Titles
FILE SEGMENT: ISRCTN
FILE SEGMENT: ClinicalTrials.gov
CLINICAL TRIAL NO.: ISRCTN30515245; ISRCTN48523317; ISRCTN67856342; NCT00091910; NCT00140010; NCT00210626; NCT00210756; NCT00260052; NCT00267007; NCT00298597; NCT00334737; NCT00336466; NCT00355095; NCT00362414; NCT00375869; NCT00413946; NCT00478517; NCT00513240; NCT00561067; NCT00589953; NCT00604630; NCT00615368; NCT00622934; NCT00626574; NCT00631202; NCT00647998; NCT00663416; NCT00697064; NCT00704652; NCT00715364; NCT00719407; NCT00737893; NCT00756249
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 17 Feb 2009
Last Updated on STN: 17 Feb 2009

AB The growth factor erythropoietin (EPO) and erythropoietin receptors (EPOR) are expressed in the nervous system. Neuronal expression of EPO and EPOR peaks during brain development and is upregulated in the adult brain after injury. Peripherally administered EPO, and at least some of its variants, cross the blood-brain barrier, stimulate neurogenesis, neuronal differentiation, and activate brain neurotrophic, anti-apoptotic, anti-oxidant and anti-inflammatory signaling. These mechanisms underlie their tissue protective effects in nervous system disorders. As the tissue protective functions of EPO can be separated from its stimulatory action on hematopoiesis, novel EPO derivatives and mimetics, such as asialo-EPO and carbamoylated EPO have been developed. While the therapeutic potential of the novel EPO derivatives continues to be characterized in preclinical studies, the experimental findings in support for the use of recombinant human (rh)EPO in human brain disease have already been translated to clinical studies in acute ischemic stroke, chronic schizophrenia, and chronic progressive multiple sclerosis. In this review article, we assess the studies on EPO and, in particular, on its structural or functional variants in experimental models of nervous system disorders, and we provide a short overview of the completed and ongoing clinical studies testing EPO as neuroprotective/neuroregenerative treatment option in neuropsychiatric disease. .COPYRGT. 2009 The American Society for Experimental NeuroTherapeutics, Inc.

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ACCESSION NUMBER: 2009381250 EMBASE
TITLE: Immunomodulation in the critically ill.
AUTHOR: Webster, N.R.; Galley, H.F.
CORPORATE SOURCE: Anaesthesia and Intensive Care, Institute of Medical Sciences, University of Aberdeen, Foresterhill, Aberdeen AB25 2ZD, United Kingdom. h.f.galley@abdn.ac.uk
AUTHOR: Webster, N. R. (correspondence)
CORPORATE SOURCE: Anaesthesia and Intensive Care, Institute of Medical Sciences, University of Aberdeen, Foresterhill, Aberdeen AB25 2ZD, United Kingdom.
SOURCE: British Journal of Anaesthesia, (July 2009) Vol. 103, No. 1, pp. 70-81.
Refs: 85
ISSN: 0007-0912; E-ISSN: 1471-6771 CODEN: BJANAD
PUBLISHER: Oxford University Press, Great Clarendon Street, Oxford, OX2 6DP, United Kingdom.
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
018 Cardiovascular Diseases and Cardiovascular Surgery
024 Anesthesiology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 2 Sep 2009
Last Updated on STN: 2 Sep 2009
AB Immunotherapy in the critically ill is an appealing notion because of the apparent abnormal immune and inflammatory responses seen in so many patients. The administration of a medication that could alter immune responses and decrease mortality in patients with sepsis could represent a 'magic bullet'. Various approaches have been tried over the last 20 yr: steroids; anti-endotoxin or anti-cytokine antibodies; cytokine receptor antagonists; and other agents with immune-modulating side-effects. However, in some respects, research along these lines has been unsuccessful or disappointing at best. The current state of knowledge is summarized with particular reference to sepsis and the acute respiratory distress syndrome.

L3 ANSWER 6 OF 24 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2009232367 EMBASE
TITLE: Ischemic neuronal damage.
AUTHOR: Taoufik, Era (correspondence); Probert, Lesley
CORPORATE SOURCE: Department of Molecular Genetics, Hellenic Pasteur Institute, 127 Vas. Sofias Avenue, 11521 Athens, Greece. etaoufik@pasteur.gr
AUTHOR: Taoufik, Era (correspondence)
CORPORATE SOURCE: Laboratory of Molecular Genetics, Hellenic Pasteur Institute, 127 Vassilissis Sofias Avenue, 11521 Athens, Greece. etaoufik@pasteur.gr
SOURCE: Current Pharmaceutical Design, (2008) Vol. 14, No. 33, pp. 3565-3573.
Refs: 124
ISSN: 1381-6128 CODEN: CPDEFP
PUBLISHER: Bentham Science Publishers B.V., P.O. Box 294, Bussum, 1400 AG, Netherlands.
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 008 Neurology and Neurosurgery
037 Drug Literature Index

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 19 Jun 2009
Last Updated on STN: 19 Jun 2009

AB Knowledge of the molecular mechanisms that underlie neuron death following stroke is important to allow the development of effective neuroprotective strategies. Since studies in human stroke are extremely limited due to the inability of collecting post mortem tissue at time points after the onset of stroke where neuronal death occurs, brain ischemia research focuses on information derived from animal models of ischemic injury. The two principal models for human stroke are induced in rodents either by global or focal ischemia. In both cases, blood flow disruptions limit the delivery of oxygen and glucose to neurons causing ATP reduction and energy depletion, initiating excitotoxic mechanisms that are deleterious for neurons. These include activation of glutamate receptors and release of excess glutamate in the extracellular space inducing neuron depolarisation and dramatic increase of intracellular calcium that in turn activates multiple intracellular death pathways. The notion that excitotoxicity leads only to neuron necrosis has been abandoned, as ultrastructural and biochemical analysis have shown signs of apoptotic and autophagic cell death in ischemic neurons and this has been further confirmed in neurons subjected to in vitro ischemia models. Both in vitro and in vivo studies, targeting a single death mechanism either by the inhibition of death-inducing molecules or the overexpression of antiapoptotic components in neurons, have shown tremendous neuroprotective potential. Despite their effectiveness in preclinical studies, a large number of neuroprotectants have failed in clinical trials for stroke suggesting that we still lack essential knowledge on the triggers and mediators of ischemic neuron death. In this review evidence will be presented on how ischemic injury occurs, what death mechanisms are activated and how these can be manipulated to induce neuroprotection. .COPYRGT. 2008 Bentham Science Publishers Ltd.

ACCESSION NUMBER: 2008215208 EMBASE
TITLE: Surgically relevant aspects of stem cell paracrine effects.
AUTHOR: Crisostomo, Paul R.; Markel, Troy A.; Wang, Yue; Meldrum, Daniel R. (correspondence)
CORPORATE SOURCE: Department of Surgery, Indiana University School of Medicine, Indianapolis, Ind, United States. dmeldrum@iupui.edu
AUTHOR: Meldrum, Daniel R. (correspondence)
CORPORATE SOURCE: Department of Physiology, Indiana University School of Medicine, Indianapolis, Ind, United States. dmeldrum@iupui.edu
AUTHOR: Meldrum, Daniel R. (correspondence)
CORPORATE SOURCE: Center for Immunobiology, Indiana University School of Medicine, Indianapolis, Ind, United States. dmeldrum@iupui.edu
SOURCE: Surgery, (May 2008) Vol. 143, No. 5, pp. 577-581.
Refs: 9
ISSN: 0039-6060 CODEN: SURGAZ
PUBLISHER IDENT.: S 0039-6060(07)00740-4
COUNTRY: United States
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 021 Developmental Biology and Teratology
025 Hematology
029 Clinical and Experimental Biochemistry
LANGUAGE: English
ENTRY DATE: Entered STN: 3 Jun 2008
Last Updated on STN: 3 Jun 2008

L3 ANSWER 8 OF 24 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2007619032 EMBASE
TITLE: Stem cells and neurological diseases.
AUTHOR: Hess, D.C. (correspondence); Borlongan, C.V.
CORPORATE SOURCE: Department of Neurology, Medical College of Georgia, Augusta, GA 30912, United States. dhess@mail.mcg.edu
AUTHOR: Borlongan, C.V.
CORPORATE SOURCE: Medical Research Service, VA Medical Center, Augusta, GA 30912, United States.
SOURCE: Cell Proliferation, (Feb 2008) Vol. 41, No. S1, pp. 94-114. Refs: 145
ISSN: 0960-7722; E-ISSN: 1365-2184 CODEN: CPROEM
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Conference Article; (Conference paper)
FILE SEGMENT: 021 Developmental Biology and Teratology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
039 Pharmacy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 Jan 2008
Last Updated on STN: 28 Jan 2008

AB Cells of the central nervous system were once thought to be incapable of regeneration. This dogma has been challenged in the last decade with studies showing new, migrating stem cells in the brain in many rodent injury models and findings of new neurones in the human hippocampus in adults. Moreover, there are reports of bone marrow-derived cells developing neuronal and vascular phenotypes and aiding in repair of injured brain. These findings have fuelled excitement and interest in regenerative medicine for neurological diseases, arguably the most difficult diseases to treat. There are numerous proposed regenerative approaches to neurological diseases. These include cell therapy approaches in which cells are delivered intracerebrally or are infused by an intravenous or intra-arterial route; stem cell mobilization approaches in which endogenous stem and progenitor cells are mobilized by cytokines such as granulocyte colony stimulatory factor (GCSF) or chemokines such as SDF-1; trophic and growth factor support, such as delivering brain-derived neurotrophic factor (BDNF) or glial-derived neurotrophic factor (GDNF) into the brain to support injured neurones; these approaches may be used together to maximize recovery. While initially, it was thought that cell therapy might work by a 'cell replacement' mechanism, a large body of evidence is emerging that cell therapy works by providing trophic or 'chaperone' support to the injured tissue and brain. Angiogenesis and neurogenesis are coupled in the brain. Increasing angiogenesis with adult stem cell approaches in rodent models of stroke leads to preservation of neurones and improved functional outcome. A number of stem and progenitor cell types has been proposed as therapy for neurological disease ranging from neural stem cells to bone marrow derived stem cells to embryonic stem cells. Any cell therapy approach to neurological disease will have to be scalable and easily commercialized if it will have the necessary impact on public health. Currently, bone marrow-derived cell populations such as the marrow stromal cell, multipotential progenitor cells, umbilical cord stem cells and neural stem cells meet these criteria the best. Of great clinical significance, initial evidence suggests these cell types may be delivered by an allogeneic approach, so strict tissue matching may not be necessary. The most immediate impact on patients will be achieved by making use of the trophic support capability of cell therapy and not by a cell replacement mechanism. .COPYRGT. 2008 The Authors.

L3 ANSWER 9 OF 24 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN
DUPLICATE 1
ACCESSION NUMBER: 2008:104438 BIOSIS
DOCUMENT NUMBER: PREV200800103257
TITLE: Simultaneous detections of 27 cytokines during cerebral wound healing by multiplexed bead-based immunoassay for wound age estimation.
AUTHOR(S): Takamiya, Masataka [Reprint Author]; Fujita, Sachiko; Saigusa, Kiyoshi; Aoki, Yasuhiro
CORPORATE SOURCE: Iwate Med Univ, Sch Med, Dept Legal Med, 19-1 Uchimaru, Morioka, Iwate 020, Japan
mtakamiy@iwate-med.ac.jp
SOURCE: Journal of Neurotrauma, (DEC 2007) Vol. 24, No. 12, pp. 1833-1844.
ISSN: 0897-7151.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 6 Feb 2008
Last Updated on STN: 6 Feb 2008
AB Quantification of 27 cytokines following cerebral wounding was performed for wound age estimation. The cytokines evaluated included interleukin (IL)-1 alpha, IL-1 beta, IL-2, IL-3, IL-4, IL-5, IL-6, IL-10, IL-12 p40, IL-12 p70, IL-15, IL-17, IL-18, basic fibroblast growth factor (bFGF), granulocyte-colony stimulating factor (G-CSF), granulocyte macrophage-colony stimulating factor (GM-CSF), Interferon-gamma (IFN-gamma), keratinocyte derived cytokine (KC), leukemia inhibitory factor (LIF), macrophage-colony stimulating factor (M-CSF), monokine inducible by interferon gamma (MIG), macrophage inflammatory protein (MIP)-1 alpha, MIP 2, platelet-derived growth factor BB (PDGF BB), regulated upon activation, normal T-cell expressed, and secreted (Rantes), tumor necrosis factor -alpha (TNF-alpha), and vascular endothelial growth factor (VEGF). The proliferation of glial cells as well as the infiltration of inflammatory cells were also evaluated. Although astroglia proliferated from 72 hours post-injury, inflammatory cell dynamics were generally steady. Among cytokines analyzed in the present study, IL-1 beta, IL-5, IL-6, IL-12 p40, G-CSF, IFN-gamma, KC, LIF, MIP 2, and PDGF BB increased during the early phase of cerebral wound healing, and M-CSF increased during the middle phase, while IL-15, IL-18, and MIG increased during the late phase. In contrast, IL-1 alpha, IL-10, IL-12 p70, and TNF-alpha were suppressed throughout the cerebral wound healing process. Based on our findings, quantitative cytokine analyses at the cerebral wound site may be a useful tool for wound age estimation. Further, this study suggests that multiplex data gained from the same sample using a single methodology demonstrates highly accurate cytokine interactions during the process of cerebral wound healing.

L3 ANSWER 10 OF 24 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2007304395 EMBASE
TITLE: Migration, fate and in vivo imaging of adult stem cells in the CNS.
AUTHOR: Sykove, E.; Jendelova, P.
CORPORATE SOURCE: Department of Neuroscience, Institute of Experimental Medicine ASCR, Prague, Czech Republic.
AUTHOR: Sykove, E.; Jendelova, P.
CORPORATE SOURCE: Center for Cell Therapy and Tissue Repair, Charles University, Second Medical Faculty, Prague, Czech Republic.
AUTHOR: Sykove, E.; Jendelova, P.
CORPORATE SOURCE: Department of Neuroscience, Charles University, Second Medical Faculty, Prague, Czech Republic.
AUTHOR: Sykovia, E. (correspondence)

CORPORATE SOURCE: Department of Neuroscience, Institute of Experimental Medicine ASCR, Prague, Czech Republic.
SOURCE: Cell Death and Differentiation, (Jul 2007) Vol. 14, No. 7, pp. 1336-1342.
Refs: 61
ISSN: 1350-9047; E-ISSN: 1476-5403 CODEN: CDDIEK
PUBLISHER IDENT.: 4402140
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 014 Radiology
029 Clinical and Experimental Biochemistry
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 24 Jul 2007
Last Updated on STN: 24 Jul 2007

AB Adult stem cells have been intensively studied for their potential use in cell therapies for neurodegenerative diseases, ischemia and traumatic injuries. One of the most promising cell sources for autologous cell transplantation is bone marrow, containing a heterogenous cell population that can be roughly divided into hematopoietic stem and progenitor cells and mesenchymal stem cells (MSCs). MSCs are multipotent progenitor cells that, in the case of severe tissue ischemia or damage, can be attracted to the lesion site, where they can secrete bioactive molecules, either naturally or through genetic engineering. They can also serve as vehicles for delivering therapeutic agents. Mobilized from the marrow, sorted or expanded in culture, MSCs can be delivered to the damaged site by direct or systemic application. In addition, MSCs can be labeled with superparamagnetic nanoparticles that allow in vivo cell imaging. Magnetic resonance imaging (MRI) is thus a suitable method for in vivo cell tracking of transplanted cells in the host organism. This review will focus on cell labeling for MRI and the use of MSCs in experimental and clinical studies for the treatment of brain and spinal cord injuries.

L3 ANSWER 11 OF 24 EMBASE
ACCESSION NUMBER: 2007288983 EMBASE
TITLE: Systemic complications after head injury: A clinical review.
AUTHOR: Lim, H.B.; Smith, Martin, Dr. (correspondence)
CORPORATE SOURCE: Department of Neuroanaesthesia and Neurocritical Care, The National Hospital for Neurology and Neurosurgery, Queen Square, London WC1N 3BG, United Kingdom. martin.smith@uclh.nhs.uk
SOURCE: Anaesthesia, (May 2007) Vol. 62, No. 5, pp. 474-482.
Refs: 87
ISSN: 0003-2409; E-ISSN: 1365-2044 CODEN: ANASAB
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 018 Cardiovascular Diseases and Cardiovascular Surgery
025 Hematology
037 Drug Literature Index
038 Adverse Reactions Titles
004 Microbiology: Bacteriology, Mycology, Parasitology and Virology
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 23 Jul 2007
Last Updated on STN: 23 Jul 2007

AB Non-neurological organ dysfunction is common after traumatic

brain injury and is an independent contributor to morbidity and mortality. It represents a risk factor that is potentially amenable to treatment, and early recognition and prompt intervention may improve outcome. This article reviews the current evidence for the mechanisms and treatment of non-neurological organ dysfunction after head injury. .COPYRGT. 2007 The Authors Journal compilation .COPYRGT. 2007 The Association of Anaesthetists of Great Britain and Ireland.

L3 ANSWER 12 OF 24 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2007119799 MEDLINE
DOCUMENT NUMBER: PubMed ID: 17316158
TITLE: Thiopental-induced neutropenia in two patients with severe head trauma.
AUTHOR: Frenette Anne Julie; Perreault Marc M; Lam Stefanie; Williamson David R
CORPORATE SOURCE: Department of Pharmacy Services, Hopital du Sacre-Coeur de Montreal, Quebec, Canada.. anjue@yahoo.com
SOURCE: Pharmacotherapy, (2007 Mar) Vol. 27, No. 3, pp. 464-71. Journal code: 8111305. ISSN: 0277-0008.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CASE REPORTS) Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200705
ENTRY DATE: Entered STN: 27 Feb 2007
Last Updated on STN: 9 May 2007
Entered Medline: 8 May 2007
AB Thiopental has been used for decades in the treatment of refractory intracranial hypertension in patients with traumatic and nontraumatic head injuries. Commonly reported adverse effects include hypotension, hypokalemia, respiratory complications, and hepatic dysfunction. Neutropenia has rarely been reported as an adverse effect of thiopental. We witnessed probable thiopental-induced neutropenia in two patients with traumatic brain injuries who developed increased intracranial hypertension that was refractory to standard therapy. Based on a MEDLINE search of published case reports and literature, we propose two mechanisms by which thiopental-related neutropenia might be explained. The first is inhibition of inflammatory mediator nuclear factor-kappa B (NF-kappa B), leading to granulocyte apoptosis. The second mechanism involves inhibition of calcineurin. Although the precise link between these two mechanisms has not been elucidated, calcineurin is known to regulate NF-kappa B activity. Development of neutropenia does not appear to be correlated with time but may correlate with plasma concentrations of thiopental. The optimum management of drug-induced neutropenia is unclear. The decision to discontinue thiopental in patients who develop neutropenia should be made by weighing the risks versus benefits. Broad-spectrum antibiotics may be required in the presence of fever. The role of hematopoietic growth factors such as granulocyte colony-stimulating factor is not yet defined. Given the adverse infectious consequences of neutropenia, it is essential to closely monitor neutrophil counts in patients receiving thiopental.

L3 ANSWER 13 OF 24 CAPLUS COPYRIGHT 2009 ACS on STN
ACCESSION NUMBER: 2007:241437 CAPLUS
DOCUMENT NUMBER: 147:1751
TITLE: Transient neuroprotection by minocycline following traumatic brain injury is associated with attenuated microglial activation but no changes in cell apoptosis or neutrophil infiltration

AUTHOR(S): Bye, Nicole; Habgood, Mark D.; Callaway, Jennifer K.; Malakooti, Nakisa; Potter, Ann; Kossmann, Thomas; Morganti-Kossmann, M. Cristina
CORPORATE SOURCE: National Trauma Research Institute and Department of Trauma Surgery, Alfred Hospital, Victoria, Australia
SOURCE: Experimental Neurology (2007), 204(1), 220-233
CODEN: EXNEAC; ISSN: 0014-4886
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cerebral inflammation and apoptotic cell death are two processes implicated in the progressive tissue damage that occurs following traumatic brain injury (TBI), and strategies to inhibit one or both of these pathways are being investigated as potential therapies for TBI patients. The tetracycline derivative minocycline was therapeutically effective in various models of central nervous system injury and disease, via mechanisms involving suppression of inflammation and apoptosis. We therefore investigated the effect of minocycline in TBI using a closed head injury model. Following TBI, mice were treated with minocycline or vehicle, and the effect on neurol. outcome, lesion volume, inflammation and apoptosis was evaluated for up to 7 days. Our results show that while minocycline decreases lesion volume and improves neurol. outcome at 1 day post-trauma, this response is not maintained at 4 days. The early beneficial effect is likely not due to anti-apoptotic mechanisms, as the d. of apoptotic cells is not affected at either time-point. However, protection by minocycline is associated with a selective anti-inflammatory response, in that microglial activation and interleukin-1β expression are reduced, while neutrophil infiltration and expression of multiple cytokines are not affected. These findings demonstrate that further studies on minocycline in TBI are necessary in order to consider it as a novel therapy for brain-injured patients.

OS.CITING REF COUNT: 23 THERE ARE 23 CAPLUS RECORDS THAT CITE THIS RECORD (23 CITINGS)
REFERENCE COUNT: 55 THERE ARE 55 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L3 ANSWER 14 OF 24 EMBASE
ACCESSION NUMBER: 2007260686 EMBASE
TITLE: MRI of mouse models of neurological disorders.
AUTHOR: Anderson, Stasia A. (correspondence)
CORPORATE SOURCE: Animal MRI/Imaging Core, National Heart Lung and Blood Institute, NIH, 10 Center Drive, Bethesda, MD 20892, United States. andersos1@nhlbi.nih.gov
AUTHOR: Frank, Joseph A.
CORPORATE SOURCE: Experimental Neuroimaging Section, Laboratory of Diagnostic Radiology Research, NIH, 10 Center Drive, Bethesda, MD 20892, United States. sanderso@helix.nih.gov
AUTHOR: Anderson, Stasia A. (correspondence)
CORPORATE SOURCE: NHLBI, Animal MRI/Imaging Core, National Institutes of Health, 10 Center Drive, Bethesda, MD 20892, United States. andersos1@nhlbi.nih.gov
SOURCE: NMR in Biomedicine, (May 2007) Vol. 20, No. 3, pp. 200-215.
Refs: 96
ISSN: 0952-3480; E-ISSN: 1099-1492 CODEN: NMRBEF
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 014 Radiology
016 Cancer
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery

LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 2 Jul 2007
Last Updated on STN: 2 Jul 2007

AB MRI has contributed to significant advances in the understanding of neurological diseases in humans. It has also been used to evaluate the spectrum of mouse models spanning from developmental abnormalities during embryogenesis, evaluation of transgenic and knockout models, through various neurological diseases such as stroke, tumors, degenerative and inflammatory diseases. The MRI techniques used clinically are technically more challenging in the mouse because of the size of the brain; however, mouse imaging provides researchers with the ability to explore cellular and molecular imaging that one day may translate into clinical practice. This article presents an overview of the use of MRI in mouse models of a variety of neurological disorders and a brief review of cellular imaging using magnetically tagged cells in the mouse central nervous system.

L3 ANSWER 15 OF 24 EMBASE

ACCESSION NUMBER: 2006582146 EMBASE
TITLE: Differential regulation of blood-brain barrier permeability in brain trauma and pneumococcal meningitis-role of Src kinases.
AUTHOR: Paul, Robert (correspondence); Angele, Barbara; Popp, Bernadette; Klein, Matthias; Riedel, Eva; Pfister, Hans-Walter; Koedel, Uwe
CORPORATE SOURCE: Department of Neurology, Klinikum Grosshadern, Ludwig-Maximilians University, Marchioninistr. 15, D-81377 Munich, Germany. Robert.Paul@med.uni-muenchen.de
SOURCE: Experimental Neurology, (Jan 2007) Vol. 203, No. 1, pp. 158-167.
Refs: 43
ISSN: 0014-4886; E-ISSN: 1090-2430 CODEN: EXNEAC
PUBLISHER IDENT.: S 0014-4886(06)00470-5
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 026 Immunology, Serology and Transplantation
029 Clinical and Experimental Biochemistry
005 General Pathology and Pathological Anatomy
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 28 Dec 2006
Last Updated on STN: 28 Dec 2006

AB Increased vascular permeability causing vasogenic brain edema is characteristic for many acute neurological diseases such as stroke, brain trauma, and meningitis. Src family kinases, especially c-Src, play an important role in regulating blood-brain barrier permeability in response to VEGF, but also mediate leukocyte function and cytokine signalling. Here we demonstrate that pharmacological inhibition of Src or c-Src deficiency does not influence cerebrospinal fluid (CSF) pleocytosis, brain edema formation, and bacterial outgrowth during experimental pneumococcal meningitis despite the increased cerebral expression of inflammatory chemokines, such as IL-6, CCL-9, CXCL-1, CXCL-2 and G-CSF as determined by protein array analysis. In contrast, inhibition of Src significantly reduced brain edema formation, lesion volume, and clinical worsening in cold-induced brain injury without decreasing cytokine/chemokine expression. While brain trauma was associated with increased cerebral VEGF formation, VEGF levels significantly declined during pneumococcal meningitis. Therefore, we conclude that in brain trauma blood-brain barrier tightness is regulated by the VEGF/Src pathway whereas c-Src does not influence brain edema

formation and leukocyte function during bacterial meningitis. .COPYRGT. 2006 Elsevier Inc. All rights reserved.

L3 ANSWER 16 OF 24 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 2007260606 EMBASE
TITLE: Bone marrow-derived stem cells in neurological diseases: Stones or masons?.
AUTHOR: Mezey, Eva (correspondence)
CORPORATE SOURCE: 49 Convent Drive, Bethesda, MD 20892, United States. mezeye@mail.nih.gov
SOURCE: Regenerative Medicine, (Jan 2007) Vol. 2, No. 1, pp. 37-49. Refs: 103
ISSN: 1746-0751
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review; (Review)
FILE SEGMENT: 016 Cancer
021 Developmental Biology and Teratology
029 Clinical and Experimental Biochemistry
030 Clinical and Experimental Pharmacology
037 Drug Literature Index
008 Neurology and Neurosurgery
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 2 Jul 2007
Last Updated on STN: 2 Jul 2007

AB In spite of the commonly held belief that 'the brain does not regenerate', it is now accepted that postnatal neurogenesis does occur. Thus, one wonders whether cellular-replacement therapy might be used to heal the brain in diseases caused by neuronal cell loss. The existence of neural stem cells has been demonstrated by many scientists and is now generally accepted. The exact role of these cells, how their numbers are regulated and how they participate in CNS and spinal cord regeneration in postnatal life are still not well known. There are many reviews summarizing work on these cells; consequently, I will focus instead on other cells that may participate in postnatal neurogenesis: bone marrow-derived stem cells. The possibility that bone marrow-derived stem cells populate the CNS and differentiate into various neural elements is certainly not universally accepted. .COPYRGT. 2007 Future Medicine Ltd.

L3 ANSWER 17 OF 24 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 2006:448251 BIOSIS
DOCUMENT NUMBER: PREV200600448349
TITLE: Granulocyte colony-stimulating factor does not affect contusion size, brain edema or cerebrospinal fluid glutamate concentrations in rats following controlled cortical impact.
AUTHOR(S): Sakowitz, O. W. [Reprint Author]; Schardt, C.; Neher, M.; Stover, J. F.; Unterberg, A. W.; Kiening, K. L.
CORPORATE SOURCE: Univ Heidelberg, Dept Neurosurg, Neuenheimer Feld 400, D-69120 Heidelberg, Germany
SOURCE: Hoff, JT [Editor]; Keep, RF [Editor]; Xi, G [Editor]; Hua, Y [Editor]. Acta Neurochir. Suppl., (2006) pp. 139-143. Brain Edema XIII.
Publisher: SPRINGER-VERLAG WIEN, SACHSENPLATZ 4-6, A-1201 VIENNA, AUSTRIA. Series: ACTA NEUROCHIRURGICA SUPPLEMENTA.
Meeting Info.: 13th International Symposium on Brain Edema and Tissue Injury. Ann Arbor, MI, USA. June 01 -03, 2005.
CODEN: ANCSBM. ISSN: 0065-1419. ISBN: 3-211-30712-5(H).
DOCUMENT TYPE: Book; (Book Chapter)
Conference; (Meeting)

LANGUAGE: English
ENTRY DATE: Entered STN: 13 Sep 2006
Last Updated on STN: 13 Sep 2006
AB Introduction. Granulocyte colony-stimulating factor (G-CSF) is an established treatment in the neutropenic host. Usage in head-injured patients at risk for infection may aggravate brain damage. In contrast, evidence of G-CSF neuroprotective effects has been reported in rodent models of focal cerebral ischemia. We investigated effects of G-CSF in acute focal traumatic brain injury (TBI) in rats.Methods. Thirty-six male Sprague-Dawley rats were anesthetized with 1.2% to 2.0% isoflurane and subjected to controlled cortical impact injury (CCII). Thirty minutes following CCII, either vehicle or G-CSF was administered intravenously. Animals were sacrificed 24 hours following CCII Glutamate concentrations were determined in cisternal cerebrospinal fluid (CSF). Brain edema was assessed gravimetrically. Contusion size was estimated by 2,3,5-triphenyltetrazolium chloride staining and volumetric analysis. Results. Dose-dependent leukocytosis was induced by infusion of G-CSF. Physiological variables were unaffected. Water content of the traumatized hemisphere and CSF glutamate concentrations were unchanged by treatment. Contusion volume was similar in all groups.Conclusions. A single injection of G-CSF did not influence cortical contusion volume, brain edema, or glutamate concentrations in CSF determined 24 hours following CCII in rats. G-CSF, administered 30 minutes following experimental TBI, failed to exert neuroprotective effects.

L3 ANSWER 18 OF 24 MEDLINE on STN
ACCESSION NUMBER: 2006247656 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16671442
TITLE: Granulocyte colony-stimulating factor does not affect contusion size, brain edema or cerebrospinal fluid glutamate concentrations in rats following controlled cortical impact.
AUTHOR: Sakowitz O W; Schardt C; Neher M; Stover J F; Unterberg A W; Kiening K L
CORPORATE SOURCE: Department of Neurosurgery, University of Heidelberg, Heidelberg, Germany.. oliver.sakowitz@med.uni-heidelberg.de
SOURCE: Acta neurochirurgica. Supplement, (2006) Vol. 96, pp. 139-43.
Journal code: 100962752. ISSN: 0065-1419.
PUB. COUNTRY: Austria
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200606
ENTRY DATE: Entered STN: 5 May 2006
Last Updated on STN: 13 Jun 2006
Entered Medline: 12 Jun 2006
AB INTRODUCTION: Granulocyte colony-stimulating factor (G-CSF) is an established treatment in the neutropenic host. Usage in head-injured patients at risk for infection may aggravate brain damage. In contrast, evidence of G-CSF neuroprotective effects has been reported in rodent models of focal cerebral ischemia. We investigated effects of G-CSF in acute focal traumatic brain injury (TBI) in rats. METHODS: Thirty-six male Sprague-Dawley rats were anesthetized with 1.2%) to 2.0% isoflurane and subjected to controlled cortical impact injury (CCII). Thirty minutes following CCII, either vehicle or G-CSF was administered intravenously. Animals were sacrificed 24 hours following CCII.

Glutamate concentrations were determined in cisternal cerebrospinal fluid (CSF). Brain edema was assessed gravimetrically. Contusion size was estimated by 2,3,5-triphenyltetrazolium chloride staining and volumetric analysis. RESULTS: Dose-dependent leukocytosis was induced by infusion of G-CSF. Physiological variables were unaffected. Water content of the traumatized hemisphere and CSF glutamate concentrations were unchanged by treatment. Contusion volume was similar in all groups. CONCLUSIONS: A single injection of G-CSF did not influence cortical contusion volume, brain edema, or glutamate concentrations in CSF determined 24 hours following CCII in rats. G-CSF, administered 30 minutes following experimental TBI, failed to exert neuroprotective effects.

L3 ANSWER 19 OF 24 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2005211492 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15845082
TITLE: The neuroprotective effect of progesterone after traumatic brain injury in male mice is independent of both the inflammatory response and growth factor expression.
AUTHOR: Jones Nigel C; Constantin Despina; Prior Malcolm J W; Morris Peter G; Marsden Charles A; Murphy Sean
CORPORATE SOURCE: Institute of Cell Signalling, University of Nottingham, Clifton Blvd., Nottingham NG7 2UH, UK.
SOURCE: The European journal of neuroscience, (2005 Mar) Vol. 21, No. 6, pp. 1547-54.
Journal code: 8918110. ISSN: 0953-816X.
PUB. COUNTRY: France
DOCUMENT TYPE: (COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200507
ENTRY DATE: Entered STN: 23 Apr 2005
Last Updated on STN: 27 Jul 2005
Entered Medline: 26 Jul 2005
AB Previous studies suggest that progesterone may possess neuroprotective properties after traumatic insult but, with the exception of reduced formation of cerebral oedema, limited experimental evidence has been presented to support this claim. In the present study we focused on the effect of progesterone treatment on structural and functional deficits in an experimental model of traumatic brain injury. Female mice exhibited significantly (P = 0.0445) reduced lesion volumes compared with males after aseptic cryogenic cerebral injury (ACI), suggesting that female sex steroids provide protection against this injury. In male mice, progesterone treatment after injury (three intraperitoneal doses of 8 mg/kg) reduced lesion volume (P = 0.0429) and improved performance in a spatial cognitive task (Morris water maze; P = 0.0014). However, progesterone had no demonstrable effect on the formation of oedema as measured using T2-weighted magnetic resonance imaging, nor did it affect brain water content. The pro-inflammatory cytokines TNF-alpha and IL-1beta, and growth factors BDNF and G-CSF, were all strongly transcriptionally activated after ACI. However, progesterone administration did not affect expression of these genes. This study provides strong evidence that progesterone possesses neuroprotective properties in a mouse model of traumatic brain injury, but suggests that the steroid achieves this effect through mechanism(s) independent of the inflammatory response or growth factor up-regulation.

L3 ANSWER 20 OF 24 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 2005013677 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15640641
TITLE: Effect of granulocyte colony-stimulating factor on functional and histopathologic outcome after traumatic brain injury in mice.
AUTHOR: Sheibani Negar; Grabowski Eric F; Schoenfeld David A; Whalen Michael J
CORPORATE SOURCE: Department of Pediatric Critical Care Medicine, The Massachusetts General Hospital and Harvard Medical School, Boston, MA, USA.
CONTRACT NUMBER: 5K08NS41969 (United States NINDS NIH HHS)
SOURCE: Critical care medicine, (2004 Nov) Vol. 32, No. 11, pp. 2274-8.
Journal code: 0355501. ISSN: 0090-3493.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE) (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200502
ENTRY DATE: Entered STN: 11 Jan 2005
Last Updated on STN: 4 Feb 2005
Entered Medline: 3 Feb 2005

AB OBJECTIVE: Granulocyte colony-stimulating factor has been used to reduce the risk of sepsis in patients with traumatic brain injury. However, granulocyte colony-stimulating factor exerts potent pro- and anti-inflammatory effects that could influence secondary injury, and outcome, after traumatic brain injury. Our objective was to determine the effect of granulocyte colony-stimulating factor on histopathologic, motor, and cognitive outcome after experimental traumatic brain injury in mice. DESIGN: Experimental study. SETTING: Research laboratory at the Massachusetts General Hospital, Boston, MA. SUBJECTS: Forty-eight adult male C57Bl/6 mice. INTERVENTIONS: Mice (8 wks of age, n = 16/group) were administered granulocyte colony-stimulating factor or saline subcutaneously twice per day for 7 days after controlled cortical impact or sham injury (n = 16). Absolute neutrophil counts, motor function, Morris water maze performance, and lesion volume were determined after controlled cortical impact or sham injury. MEASUREMENTS AND MAIN RESULTS: At the time of controlled cortical impact, body weight, brain and body temperature, and systemic absolute neutrophil counts did not differ between groups. Compared with control, systemic absolute neutrophil count was increased more than ten-fold in granulocyte colony-stimulating factor-treated mice on posttrauma days 2 and 7 ($p < .05$, repeated-measures analysis of variance) but did not differ between groups by day 14. There were no differences between groups in tests of motor function or histopathologic outcome. However, compared with control, mice given granulocyte colony-stimulating factor had improved Morris water maze performance after controlled cortical impact ($p < .05$, repeated-measures analysis of variance) but not sham injury. CONCLUSIONS: The data suggest a small beneficial effect of granulocyte colony-stimulating factor on functional outcome after traumatic brain injury in adult mice but do not show differences in histopathology or motor outcome between treated and control groups.

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ACCESSION NUMBER: 2003:550086 BIOSIS
DOCUMENT NUMBER: PREV200300538461
TITLE: Sepsis and hypothermia: Call in the granulocytes?.
AUTHOR(S): Gropper, Michael A. [Reprint Author]
CORPORATE SOURCE: University of California, San Francisco, CA, USA
gropperm@anesthesia.ucsf.edu
SOURCE: Anesthesiology (Hagerstown), (November 2003) Vol. 99, No. 5, pp. 1041-1043. print.
ISSN: 0003-3022 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 19 Nov 2003
Last Updated on STN: 19 Nov 2003

L3 ANSWER 22 OF 24 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2001052522 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11098978
TITLE: Effect of neutropenia and granulocyte colony stimulating factor -induced neutrophilia on blood-brain barrier permeability and brain edema after traumatic brain injury in rats.
AUTHOR: Whalen M J; Carlos T M; Wisniewski S R; Clark R S; Mellick J A; Marion D W; Kochanek P M
CORPORATE SOURCE: Department of Anesthesiology/Critical Care Medicine, University of Pittsburgh, the Safar Center for Resuscitation Research, PA 15260, USA.
CONTRACT NUMBER: NS30318 (United States NINDS NIH HHS)
SOURCE: Critical care medicine, (2000 Nov) Vol. 28, No. 11, pp. 3710-7.
Journal code: 0355501. ISSN: 0090-3493.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
(RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200012
ENTRY DATE: Entered STN: 22 Mar 2001
Last Updated on STN: 22 Mar 2001
Entered Medline: 11 Dec 2000
AB OBJECTIVE: Granulocyte colony stimulating factor (GCSF) has been used to increase systemic absolute neutrophil count (ANC) in patients with severe traumatic brain injury to reduce nosocomial infection risk. However, the effect of increasing systemic ANC on the pathogenesis of experimental traumatic brain injury has not been studied. Thus, we evaluated the effect of systemic ANC on blood-brain barrier (BBB) damage and brain edema after traumatic brain injury in rats. DESIGN: Experimental study. SETTING: Research laboratory at the University of Pittsburgh, PA. SUBJECTS: Forty-three adult male Sprague-Dawley rats. INTERVENTIONS: Protocol I: rats were randomized to receive either vinblastine sulfate to reduce ANC, GCSF to increase ANC, or saline before controlled cortical impact (CCI) of moderate overall severity. Evans blue was used to assess BBB damage at 4-24 hrs after CCI. Protocol II: rats received GCSF or saline before CCI. Brain edema was estimated at 24 hrs using wet - dry) / wet weight method. Protocol III: rats received GCSF or saline before CCI. Brain neutrophil accumulation was estimated at 24 hrs using a myeloperoxidase assay. MEASUREMENTS AND MAIN RESULTS: Physiologic variables were controlled before CCI was maintained at normal in all animals before traumatic brain injury. No

rats were anemic, hypoglycemic, or hypotensive before CCI. Protocol I: compared with control, systemic ANC decreased in vinblastine-treated rats and increased in GCSF-treated rats. BBB damage correlated with systemic ANC. Protocol II: mean systemic ANC before traumatic brain injury increased 15-fold in rats given GCSF vs. control; however no difference in brain edema was observed at 24 hrs after injury between groups. Protocol III: median systemic ANC at the time of CCI was increased ten-fold in rats given GCSF vs. control. No difference in brain myeloperoxidase activity 24 hrs after CCI was observed in rats treated with GCSF vs. control. CONCLUSIONS: Systemic ANC influences BBB damage after traumatic brain injury produced by CCI. Because BBB damage and brain edema are discordant, mechanisms other than BBB damage likely predominate in the pathogenesis of brain edema after contusion. The implications of increased BBB permeability with the administration of GCSF in our model remains to be determined. Increasing systemic ANC before CCI with GCSF administration does not increase posttraumatic brain neutrophil accumulation or brain edema after CCI in rats. The finding that neutrophil infiltration is not enhanced by systemic neutrophilia suggests that the ability of GCSF-stimulated neutrophils to migrate into injured tissue may be impaired. Further studies are needed to evaluate the effects of GCSF administration on secondary injury and functional outcome in experimental models of traumatic brain injury.

L3 ANSWER 23 OF 24 BIOSIS COPYRIGHT (c) 2009 The Thomson Corporation on STN

ACCESSION NUMBER: 2000:197816 BIOSIS
DOCUMENT NUMBER: PREV200000197816
TITLE: Clinical phase II evaluation of the combination therapy with docetaxel and epidoxorubicin in the neoadjuvant, cytostatic treatment on patients with primary breast cancer (T1-4, N0-2, M0).
AUTHOR(S): Wenzel, Catharina; Schmidinger, Manuela; Locker, Gottfried J.; Taucher, Susanne; Gnant, Michael; Jakesz, Raimund; Steger, Guenther G. [Reprint author]
CORPORATE SOURCE: Klinische Abteilung fuer Onkologie, Universitaetsklinik fuer Innere Medizin I, Waehringer Guertel 18-20, A-1090, Wien, Austria
SOURCE: Wiener Klinische Wochenschrift, (Oct. 29, 1999) Vol. 111, No. 20, pp. 843-850. print.
CODEN: WKWOAO. ISSN: 0043-5325.
DOCUMENT TYPE: Article
LANGUAGE: German
ENTRY DATE: Entered STN: 17 May 2000
Last Updated on STN: 4 Jan 2002

AB Background: Preoperative (neo-adjuvant) chemotherapy is very effective in downstaging primary tumors and moreover is able to prevent advancing metastatic growth early in the course of the disease. Methods: We report on 38 patients with a median age of 54 years (range, 33-70 years) suffering from biopsy-proven breast cancer (T1-T4). Mastectomy had been considered the treatment of choice in all cases. The patients received 194 cycles of chemotherapy with docetaxel (75 mg/m2) and epidoxorubicin (75 mg/m2) on day 1, every 21 days, together with 30 million IU of G-CSF from days 3 to 10. Three to 8 cycles (median 5 cycles) of the treatment were administered until best response was achieved on mammography and clinical assessment. Results: The neo-adjuvant chemotherapy was well tolerated and all patients completed the treatment regimen on an out-patient basis. During 194 cycles we observed leukopenia WHO grade IV only at one occasion (0.5%). WHO-grade III toxicity consisted of leukopenia (0.5%), diarrhoea (2%), and stomatitis (0,5%). Response to treatment was present in 85%, with 4 patients (11%) experiencing a pathological complete response (pCR) of the

invasive tumor (T0: n = 2, DCIS: n = 2) and 28 patients (74%) showing a partial pathological response. In 21 patients (52%) a breast-conserving surgical procedure was possible. Summary: We conclude that neo-adjuvant treatment of primary breast cancer with docetaxel and epidoxorubicin is safe and effective. By applying more chemotherapy cycles preoperatively it might even be possible to raise the rate of pCR and prolong survival.

L3 ANSWER 24 OF 24 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 1998218516 MEDLINE
DOCUMENT NUMBER: PubMed ID: 9559614
TITLE: Effect of prophylactic administration of recombinant human granulocyte colony-stimulating factor (filgrastim) on the frequency of nosocomial infections in patients with acute traumatic brain injury or cerebral hemorrhage. The Filgrastim Study Group.
AUTHOR: Heard S O; Fink M P; Gamelli R L; Solomkin J S; Joshi M; Trask A L; Fabian T C; Hudson L D; Gerold K B; Logan E D
CORPORATE SOURCE: Department of Anesthesiology, University of Massachusetts Medical Center, Worcester 01655, USA.
SOURCE: Critical care medicine, (1998 Apr) Vol. 26, No. 4, pp. 748-54.
Journal code: 0355501. ISSN: 0090-3493.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
(CLINICAL TRIAL, PHASE II)
(COMPARATIVE STUDY)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199805
ENTRY DATE: Entered STN: 20 May 1998
Last Updated on STN: 3 Mar 2000
Entered Medline: 14 May 1998

AB OBJECTIVE: To determine whether the use of prophylactic recombinant human granulocyte colony-stimulating factor (filgrastim) reduces the frequency of nosocomial infections in patients with either acute traumatic brain injury or cerebral hemorrhage. DESIGN: Randomized, placebo-controlled, double-blind, multicenter phase II study. SETTING: Intensive care units of seven medical centers. PATIENTS: Patients with either acute traumatic brain injury or cerebral hemorrhage who were intubated within 6 hrs of admission and who were expected to be ventilated for >72 hrs. INTERVENTIONS: Patients were randomized to receive daily subcutaneous injections of placebo (n = 21) or one of two doses of filgrastim (75 microg [n = 20] or 300 microg [n = 20]) for 10 days or until the absolute neutrophil count was >75,000 cells/mm3 or until extubation. MEASUREMENTS AND MAIN RESULTS: End points included increase in absolute neutrophil count, safety of filgrastim, and frequency of nosocomial infections (pneumonia, bacteremia, and urinary tract infection). Filgrastim caused a dose-dependent increase in absolute neutrophil count. There were no differences in the frequency of pneumonia or urinary tract infection; however, there was a dose-dependent decrease in the frequency of bacteremias ($p < .05$). Adverse events were similar among the three groups. There was one case of acute respiratory distress syndrome in the placebo group. CONCLUSION: In this patient population, use of filgrastim was safe and the agent appeared to reduce the risk of primary bacteremias but had no beneficial effects on mortality, length of stay, or other nosocomial infections.

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COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
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FULL ESTIMATED COST                                    87.46      87.68

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
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     FILE 'MEDLINE, BIOSIS, CAPLUS, EMBASE' ENTERED AT 12:30:13 ON 17 DEC 2009
L1         76986 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  (G(W) CSF OR GRANULOCYTE(W
                 ) COLONY(W) STIMULATING(W) FACTOR)
L2            35 SEA FILE=MFE SPE=ON  ABB=ON  PLU=ON  L1 AND (TRAUMATIC(W)
                 BRAIN(W) INJURY)
L3            24 DUP REM L2 (11 DUPLICATES REMOVED)
                 DIS IBIB ABS L3 1-24

     FILE 'STNGUIDE' ENTERED AT 12:33:09 ON 17 DEC 2009
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                     1.12      88.80

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                     0.00      -0.82

STN INTERNATIONAL LOGOFF AT 12:42:58 ON 17 DEC 2009
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